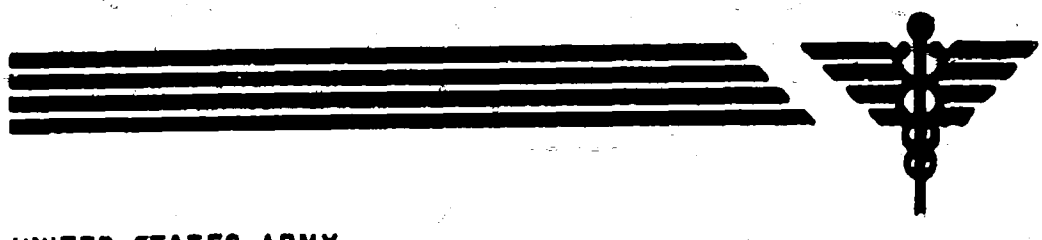
US ARMY MEDICAL RESEARCH LABORATORY

FORT KNOX, KENTUCKY

REPORT NO. 538

THE EFFECT OF AN ANALGESIC AGENT ON MUSCULAR WORK DECREMENT

Lee S. Caldwell, Ph. D.
Capt Wayne O. Evans, MSC



UNITED STATES ARMY
MEDICAL RESEARCH AND DEVELOPMENT COMMAND

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Authors

Lee S. Caldwell, Ph. D. — Psychomotor Branch, Psychology Division

Capt Wayne O. Evans, MSC (Ph. D.) — Psychomotor Branch, Psychology Division

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Project No. 6X60-01-001. Unclassified Report

UNCLASSIFIED

1. Psychopharmacology
2. Psychomotor Activity
3. Human Strength

Previous work has shown that a correlation exists between both the degree of ischemia and the intensity of pain with the strength of contraction in a tonically contracted muscle. Also, it has been demonstrated that the degree of muscle ischemia is related to the intensity of induced pain. These observations might lead to the conclusion that the pain caused by ischemia limits the endurance of the muscle contraction. An analgesic agent, known to reduce ischemic pain was shown to have no effect on the endurance of a tonic muscle contraction. This was interpreted as demonstrating a lack of causality in the relationship between pain due to ischemia and muscular work decrement.

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Psychology Division
US ARMY MEDICAL RESEARCH LABORATORY
Fort Knox, Kentucky

10 May 1962

Pharmacology of the Combat Soldier
Task 04
Internal Medicine
USAMRL Project No. 6X60-01-001

Report No. 538
USAMRL Project No. 6X60-01-001-04

ABSTRACT

THE EFFECT OF AN ANALGESIC AGENT ON MUSCULAR WORK DECREMENT

OBJECT

To determine if ischemic muscle pain and the decrement in contraction strength of a tonically contracted muscle are causally or merely correlationally related.

BACKGROUND

Previous work has shown that a correlation exists between the intensity of pain and the strength of contraction in a tonically contracted muscle. In order to determine if these relationships are causally related, human subjects were given an analgesic agent to reduce the pain aspect in the performance of an isometric muscular contraction.

RESULTS

The results showed that the administration of an analgesic had no effect on either the maximum strength or the endurance of an isometric muscular contraction.

CONCLUSION

These results are interpreted as a demonstration of the lack of a causal relationship between the decrement in work output and the pain of ischemia in a tonically contracted muscle.

APPROVED: George S. Harker

GEORGE S. HARKER, Ph. D.
Director, Psychology Division

APPROVED: Floyd A. Odell

FLOYD A. ODELL, Ph. D.
Technical Director of Research

APPROVED: Sven A. Bach

SVEN A. BACH
Colonel, MC
Commanding

THE EFFECT OF AN ANALGESIC AGENT ON MUSCULAR WORK DECREMENT

I. INTRODUCTION

Dorpat and Holmes (1) have shown that in tonically contracted muscles the time for onset and the intensity of the induced pain and the degree of ischemia are directly related to the strength of contraction. Also, it has been demonstrated that the degree of muscle ischemia was proportional to the degree of induced pain. These observations led to the conclusion that the pain associated with strong tonic contraction was due to the accumulation of noxious metabolites in the muscles made ischemic by contraction. It should follow, then, that if the relationship between ischemic pain and decrement in contraction strength is a causal one and not merely correlational, an analgesic agent such as codeine, which Hewer and Kelle (3) have shown to be effective for ischemic muscle pain, should extend the time for which a strong contraction can be maintained.

The purpose of this study was to explore a relationship between ischemic pain and decrement in muscle contraction strength to determine whether these factors are, in fact, causally related.

II. METHOD

The subjects were 14 male volunteers from the laboratory staff.

The apparatus has been previously described in detail by Caldwell (2). In brief, it consisted of an adjustable seat and foot-rest which permitted all subjects to be placed in anatomically similar positions, and an isometric handle adjustable to produce similar arm positions for all subjects. The seat was adjusted to position the approximate center of the right shoulder joint at the height of the handle, and the foot-rest was positioned to set the long axis of the thigh 20° above the horizontal and to produce a knee-angle of 150°. The handle was set to produce an elbow angle of 150°.

The output from the strain gages mounted on the handle was fed into a strain amplifier and recorded on an ink-writing oscillograph. A voltmeter with a scale marked in pounds, which was connected in parallel with the recorder, served as a display for the subject. Red and green lamps mounted on the meter were controlled by an amplifier connected in parallel with the display. The display amplifier had a

bias control which varied the switching point of a bipolar relay. Thus, the amplifier could be set for the green light to be turned on by any desired force on the handle.

Prior to the experiment, each subject was given practice in the experimental procedure and his "peak strength" was determined. To determine peak strength the subject was told that he was to pull as hard as possible for 8 seconds, and that the maximum force obtained was to be noted. The peak strength test was repeated several times until the subject's performance had stabilized. The subject's maximum output was noted. The output which was to be maintained in the subsequent endurance tests were set at 90% of the individual's peak strength. On the endurance test the display amplifier was set to turn the green light on at the appropriate output level (90% of the subject's maximum strength) and he was simply told to keep the green light on as long as possible. The trial was terminated by the experimenter when the green light remained off for as long as 3 consecutive seconds.

Each subject's strength and endurance was tested under seven conditions: a "no drug" or normal condition; four different doses of codeine sulfate (16 mg, 32 mg, 48 mg, and 64 mg); a placebo condition using 100 mg of lactose; and an "active placebo" condition with 50 mg secobarbital. The "active" placebo was used to test for a possible change in performance as a consequence of "feeling drugged." The conditions were counterbalanced to eliminate possible practice and cumulative fatigue effects. The experimental sessions were minimally 48 hours apart. The drugs and placebos were administered in identical capsules by a double blind procedure one hour before the tests. An eighth testing session was given under normal conditions as the last session to provide data for measures of test-retest reliability.

III. RESULTS AND DISCUSSION

The mean strength and endurance scores for the eight conditions are shown in Table 1.

Treatment x Subjects analyses of variance showed that the drug and placebo conditions produced no statistically significant differences in performance either in strength or endurance. The test-retest coefficients of correlation (rank-difference) for strength and endurance were .933 and .814, respectively.

These data suggest that pain is not the limiting factor in the endurance of strong tonic muscular contractions, i.e., that the

relationship between pain and strength decrement is purely correlational, not causal. The correlation noted between pain and decrement in contraction strength may result from their relationship to a third phenomenon--perhaps to the noxious metabolites (such as Lewis' Factor P) which accumulate in the muscle during sustained contraction.

When the subjects were asked why they were unable to maintain the required output, the most common report was that the pain was too intense. However, at the highest codeine dosage most of the subjects reported a generalized weakness rather than pain as the reason for the inability to continue at the specified output. The fact that codeine was given up to its maximum effective dosage (64 mg), and that opiates do effect ischemic muscle pain, adds support to the assumption that pain was decreased--at least at the higher codeine dosage.

IV. SUMMARY

Codeine sulfate was shown to have no effect on the decrement in strength of a tonically contracted muscle. This is interpreted as demonstrating a lack of causality in the relationship between ischemic pain and muscle fatigue.

V. REFERENCES

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3. Hewer, R. J. and C. A. Kelle. A method of testing analgesics in man. Lancet, 2: 683, 1948.

TABLE 1

Mean Strength and Endurance Scores

	Strength (lbs)	Endurance (secs)
Control 1	128	21.5
Control 2	133	23.5
Seconal (50 mg)	134	25.2
Lactose (100 mg)	132	23.8
Codeine (16 mg)	133	23.1
Codeine (32 mg)	134	24.6
Codeine (48 mg)	132	24.2
Codeine (64 mg)	132	23.8

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